

Amendment and Response

Serial No.: 09/877,220

Confirmation No.: 8535

Filed: June 8, 2001

For: METHODS FOR TREATING NEUROPATHOLOGICAL STATES AND NEUROGENIC
INFLAMMATORY STATES AND METHODS FOR IDENTIFYING COMPOUNDS USEFUL THEREIN

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of NR1 subunit in a cell not contacted with the compound indicates the compound alters the distribution of NR1 subunit in the cell.

21. [AMENDED] A method for identifying a compound that alters the amount of NR1 subunit in a cell, the method comprising:

- B¹
- contacting a cell with a compound;
 - activating an NMDA glutamate receptor present on the cell; and
 - detecting the amount of NR1 subunit in the cell;

wherein an alteration in the amount of NR1 subunit in the cell contacted with the compound relative to the amount of NR1 subunit in a cell not contacted with the compound indicates the compound alters the amount of NR1 subunit in the cell.

24. [AMENDED] A method for identifying a tyrosine kinase inhibitor that alters NR1 subunit distribution in a cell, the method comprising:

- B²
- contacting a cell with a tyrosine kinase inhibitor;
 - activating an NMDA glutamate receptor present in the cell; and
 - detecting the distribution of NR1 subunit in the cell, wherein an alteration in the distribution of NR1 subunit in the cell contacted with the tyrosine kinase inhibitor relative to the distribution of NR1 subunit in a cell not contacted with the tyrosine kinase inhibitor indicates the compound alters distribution of NR1 subunit.
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29. [AMENDED] A method for altering NR1 subunit distribution in a cell, the method comprising:

- B³
- contacting a cell with a compound;
 - activating an NMDA glutamate receptor present on the cell; and

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detecting the distribution of NR1 subunit in the cell, wherein the distribution of NR1 subunit in the cell contacted with the compound is altered relative to the distribution of NR1 subunit in a cell not contacted with the compound.

30. [AMENDED] The method of claim 29 wherein the amount of NR1 subunit associated with a nucleus of a cell contacted with a compound is decreased.

31. [AMENDED] The method of claim 29 wherein the amount of NR1 subunit associated with a nucleus of a cell is increased.

32. [NEW] The method of claim 20, wherein the cell is neuron.

33. [NEW] The method of claim 20, wherein the contacting a cell with a compound occurs before, during, or after activating an NMDA glutamate receptor present in the cell.

34. [NEW] The method of claim 20, wherein the alteration in the distribution of NR1 subunit in the cell is a decrease in the amount of NR1 subunit associated with the nucleus.

35. [NEW] The method of claim 20 wherein the alteration in the distribution of NR1 subunit in the cell is an increase in the amount of NR1 subunit associated with the nucleus.

36. [NEW] The method of claim 20, wherein the alteration in the distribution of NR1 subunit in the cell is a decrease in the total amount of NR1 subunit in the cell.

37. [NEW] The method of claim 20, wherein the alteration in the distribution of NR1 subunit in the cell is an increase in the total amount of NR1 subunit in the cell.

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38. [NEW] The method of claim 20, wherein the compound is selected from the group consisting of a tyrosine kinase inhibitor, a tyrosine phosphatase and a serine/threonine phosphatase.
39. [NEW] The method of claim 20, wherein the compound is selected from the group consisting of a tyrosine kinase, a tyrosine phosphatase inhibitor, and a serine/threonine phosphatase inhibitor.
40. [NEW] The method of claim 20, wherein the alteration in the distribution of NR1 subunit in the cell is associated with a decrease in the amount of phosphorylated NR1 subunit in the cell.
41. [NEW] The method of claim 20, wherein the alteration in the distribution of NR1 subunit in the cell is associated with an increase in the amount of phosphorylated NR1 subunit in the cell.
42. [NEW] The method of claim 21, wherein the cell is neuron.
43. [NEW] The method of claim 21, wherein the contacting a cell with a compound occurs before, during, or after activating an NMDA glutamate receptor present in the cell.
44. [NEW] The method of claim 21, wherein the alteration in the amount of NR1 subunit in the cell is a decrease in the total amount of NR1 subunit in the cell.
45. [NEW] The method of claim 21, wherein the alteration in the amount of NR1 subunit in the cell is an increase in the total amount of NR1 subunit in the cell.

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46. [NEW] The method of claim 21, wherein the alteration in the amount of NR1 subunit in the cell is a decrease in the amount of NR1 subunit associated with the nucleus.

47. [NEW] The method of claim 21, wherein the alteration in the amount of NR1 subunit in the cell is an increase in the amount of NR1 subunit associated with the nucleus.

48. [NEW] The method of claim 21, wherein the compound is selected from the group consisting of a tyrosine kinase inhibitor, a tyrosine phosphatase and a serine/threonine phosphatase.

49. [NEW] The method of claim 21, wherein the compound is selected from the group consisting of a tyrosine kinase, a tyrosine phosphatase inhibitor, and a serine/threonine phosphatase inhibitor.

50. [NEW] The method of claim 21, wherein the alteration in the distribution of NR1 subunit in the cell is associated with a decrease in the amount of phosphorylated NR1 subunit in the cell.

51. [NEW] The method of claim 21, wherein the alteration in the distribution of NR1 subunit in the cell is associated with an increase in the amount of phosphorylated NR1 subunit in the cell.

52. [NEW] The method of claim 24 wherein the cell is a neuron.

53. [NEW] The method of claim 24, wherein the contacting a cell with a tyrosine kinase inhibitor occurs before, during, or after activating an NMDA glutamate receptor present in the cell.

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54. [NEW] The method of claim 24, wherein the alteration in the distribution of NR1 subunit in the cell is a decrease in the amount of NR1 subunit associated with the nucleus.
55. [NEW] The method of claim 24, wherein the alteration in the distribution of NR1 subunit in the cell is an increase in the amount of NR1 subunit associated with the nucleus.
56. [NEW] The method of claim 24, wherein the alteration in the distribution of NR1 subunit in the cell is a decrease in the total amount of NR1 subunit in the cell.
57. [NEW] The method of claim 24, wherein the alteration in the distribution of NR1 subunit in the cell is an increase in the total amount of NR1 subunit in the cell.
58. [NEW] The method of claim 24, wherein the alteration in the distribution of NR1 subunit in the cell is associated with a decrease in the amount of phosphorylated NR1 subunit in the cell.
59. [NEW] The method of claim 24, wherein the alteration in the distribution of NR1 subunit in the cell is associated with an increase in the amount of phosphorylated NR1 subunit in the cell.
60. [NEW] The method of claim 29 wherein the cell is a neuron.
61. [NEW] The method of claim 29, wherein the contacting a cell with a compound occurs before, during, or after activating a NMDA glutamate receptor present in the cell.
62. [NEW] The method of claim 29, wherein the total amount of NR1 subunit in the cell is decreased.

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63. [NEW] The method of claim 29, wherein the total amount of NR1 subunit in the cell is increased.

64. [NEW] The method of claim 29, wherein the amount of phosphorylated NR1 subunit in the cell is decreased.

65. [NEW] The method of claim 29, wherein the amount of phosphorylated NR1 subunit in the cell is increased.

B³
66. [NEW] The method of claim 29, wherein the compound is selected from the group consisting of a tyrosine kinase inhibitor, a tyrosine phosphatase and a serine/threonine phosphatase.

67. [NEW] The method of claim 29, wherein the compound is selected from the group consisting of a tyrosine kinase, a tyrosine phosphatase inhibitor, and a serine/threonine phosphatase inhibitor.
